Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L5	335256	fluorescen\$3 OR luminescen\$3	US-PGPUB; USPAT;	OR	ON	2004/12/09 17:06
			EPO; JPO; DERWENT			
L6	210780	(signal OR signaling) adj (pathway OR process\$2)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/09 17:07
L7	2413	L6 near(cell\$3 OR intracellular)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/09 17:07
L8	, 0	L7 near 15	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/09 17:08
L9	77	L7 same 15	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/09 17:08
L10	7804605	method OR screen\$3	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/09 17:08
Lil.	58.	19 same L10	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/09 17:15
S1	1	("6518021").PN.	USPAT	OR	OFF	2004/12/08 17:41
S2	- 28	ole near thastrup.in.	US-PGPUB;	OR	ON	2004/12/08 17:41
			USPAT; EPO; JPO; DERWENT			
S4	16	sara near petersen.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/09 13:59
S5	8	sara near bjorn in.	US-PGPUB; USPAT; EPO; JPO;	OR	ON	2004/12/09 14:00
S6	5	kasper near almholt.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/09 14:01
S7	12	kurt near scudder in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/09 14:01
S9	1	("5874304").PN.	USPAT	OR	OFF	2004/12/09 14:44

S10	12492	(signal OR signaling) adj pathway	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/09 14:46
<b>S</b> 11	210780	(signal OR signaling) adj (pathway OR process\$2)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/09 14:53
S12	1144	S11 near cell\$3	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/09 14:48
S13	1234	S11 near \$5cell\$3	US-PGPUB; USPAT; EPO; IPO; DERWENT	OR	ON	2004/12/09 14:50.
S14	2413	S11 near(cell\$3 OR intracellular)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2004/12/09 14:54

```
L2
          164866 S (SIGNAL OR SIGNALING) (W) (PATHWAY OR PROCESS?)
L3
        12474968 S CELL? OR INTRACELLULAR
L4
           11650 S L2 (A) L3
L7
             998 S L4 (P) (METHOD OR SCREEN?)
             359 S L4 (S) (METHOD OR SCREEN?)
15 S L4 AND L1
L8
L9
L10
              15 DUP REM L9 (0 DUPLICATES REMOVED)
        1239021 S FLUORESCEN? OR LUMINESCEN?
L11
L12
             228 S L4 (P) L11
              48 S L12 (P) (METHOD OR SCREEN?)
L13
L14
              23 DUP REM L13 (25 DUPLICATES REMOVED)
L15
               2 S L14 AND PY<=1997
L14
     ANSWER 18 OF 23 CAPLUS COPYRIGHT 2004 ACS on STN
     Toyota, T.; Watanabe, A.; Shibuya, H.; Nankai, M.; Hattori, E.; Yamada,
     K.; Kurumaji, A.; Karkera, J. D.; Detera-Wadleigh, S. D.; Yoshikawa, T.
     Molecular Psychiatry (2000), 5(5), 489-494
CODEN: MOPSFQ; ISSN: 1359-4184
SO
ጥፐ
     Association study on the DUSP6 gene, an affective disorder candidate gene
     on 12q23, performed by using fluorescence resonance energy transfer-based
     melting curve analysis on the LightCycler
     The authors introduced a new genotyping method,
     fluorescence resonance energy transfer-based melting curve anal.
     on the LightCycler, for the anal. of the gene, DUSP6 (dual specificity MAP
     kinase phosphatase 6), in affective disorder patients. The DUSP6 gene is located on chromosome 12q22-23, which overlaps one of the reported bipolar disorder susceptibility loci. Because of its role in
     intracellular signaling pathways, the gene may
     be involved in the pathogenesis of affective disorders not only on the
     basis of its position but also of its function. The authors performed
     association anal. using a T>G polymorphism that gives rise to a missense
     mutation (Leul14Val). No evidence for a significant disease-causing
     effect was found in Japanese unipolars and bipolars, when compared with
     controls. More importantly, this study demonstrates that melting curve
     anal. on the LightCycler is an accurate, rapid and robust method
     for discriminating genotypes from biallelic markers. This strategy has
     the potential for use in high throughput scanning for and genotyping of
     single nucleotide polymorphisms (SNPs).
     Kovala A.T.; Harvey K.A.; McGlynn P.; Boguslawski G.; Garcia J.G.N.;
     English D.
SO
     FASEB Journal, (2000) 14/15 (2486-2494).
     Refs: 33
     ISSN: 0892-6638 CODEN: FAJOEC
TΤ
     High-efficiency transient transfection of endothelial cells for functional
     analvsis.
     The definition of signaling pathways in endothelial cells has been
     hampered by the difficulty of transiently transfecting these cells with
     high efficiency. This investigation was undertaken to develop an efficient
     technique for the transfection of endothelial cells for functional
     analyses. Cells cotransfected with plasmid expressing green fluorescent protein (GFP) and the plasmid of interest were
     isolated by fluorescence-activated cell sorting (FACS) based on
     GFP expression. In the sorted cell population, a 2.5-fold enhancement in
     the number of cells expressing the gene of interest was observed, as
```

confirmed by FACS analysis and Western blotting. Sorted cells retained functional properties, as demonstrated by chemotaxis to the agonist sphingosine 1-phosphate (SPP). To demonstrate the usefulness of this

encoding GFP and the carboxyl-terminal domain of the <SYM98>-adrenergic receptor kinase (<SYM98>ARKct), which inhibits signaling through the <SYM98><SYM103> dimer of heterotrimeric G-proteins. SPP-induced chemotaxis

demonstrating that chemotaxis was driven by a <SYM98><SYM103>-dependent pathway. However, no significant inhibition was observed in cells

transfected with <SYM98>ARKct but not enriched by sorting. Thus, we have

the elucidation of crucial mechanisms of endothelial cell activation and

in sorted cells coexpressing <SYM98>ARKct was inhibited by 80%,

developed a method for enriching transfected cells that allows

function. This method should find wide applicability in studies

method for defining cellular signaling

pathways, cells were cotransfected with plasmids

designed to define pathways responsible for regulation of motility and other functions in these dynamic cells.

- AU Fan, Li [Reprint Author]; Rivard, Georges E. [Reprint Author]; Yotov, Wagner V. [Reprint Author]; Gobeil, Fernand Jr. [Reprint Author]; Chemtob, Sylvain [Reprint Author]
- SO Blood, (November 16 2002) Vol. 100, No. 11, pp. Abstract No. 1893. print. Meeting Info.: 44th Annual Meeting of the American Society of Hematology. Philadelphia, PA, USA. December 06-10, 2002. American Society of Hematology. CODEN: BLOOAW. ISSN: 0006-4971.
- TI Induction of Cellular Translocation of Green Fluorescent Protein Tagged PAR-2 Receptor by FVIIa.
- It is still uncertain whether FVIIa-tissue factor (FVIIa/TF) complex can induce cellular signaling pathway through interaction with PAR-2 receptor. In order to obtain direct evidence, we utilized green fluorescent protein (GFP) tagged PAR-2 receptor as a marker to monitor its cellular translocation when it is activated. Briefly, both TF and GFP tagged PAR-2 receptor (at 18:1 ratio) were transiently transfected into the embryonic human kidney BOSC23 cell line endogenously expressing low level of PAR-2 receptor and TF. After 48 hours expression, living cells were monitored with a fluorescence microscope equipped with a video camera. FVIIa, PAR-2 agonist SLIGKV-NH2, or trypsin were added to the cell culture containing  $0.1\overline{\$}$  BSA . Photos were taken at 1, 20, 40 and 60 min after addition of above substances. Results showed that GFP-tagged PAR-2 receptor migrated from cell surface to the cell center upon addition of 100 nM of FVIIa, as well as 100 uM of PAR-2 agonist or 10 nM of trypsin. This suggests that the FVIIa/TF complex can activate cellular translocation of PAR-2 receptor. The effect of FVIIa, PAR-2 agonist, trypsin and thrombin on calcium mobilization was also investigated on BOSC23 cell line using Fura-2/AM method. The transfection of both PAR-2 receptor and TF plasmids produced four cell types, BOSC23, BOSC23TF, BOSC23PAR-2 and BOSC23TF + PAR-2. Results showed that FVIIa induced a higher transient Ca2+ signal in BOSC23TF + PAR-2 than that in BOSC23TF and BOSC23PAR-2. No detectable signal was observed on the non-transfected BOSC23 cell type. Compared to the strong signal induced in all cell types by PAR-2 agonist, trypsin, and thrombin, the signal induced by FVIIa was relatively weak, delayed and long-lasting. We conclude that FVIIa/TF complex may activate PAR-2 receptor by a different mechanism than the one used by PAR-2 agonist and trypsin.
- AU Umezawa Yoshio
- SO Journal of biotechnology, (2002 Feb) 82 (4) 357-70. Ref: 17 Journal code: 8411927. ISSN: 0168-1656.
- Assay and screening methods for bioactive substances based on cellular signaling pathways.
- Assay and screening methods for bioactive substances AB based on cellular signaling pathways are presented. Examples include: (1) intracellular protein phosphorylation and protein-protein interaction, (1-i) a new assay method for evaluating chemical selectivity of agonists for insulin signaling pathways based on agonist-induced phosphorylation of a target peptide, (1-ii) an SPR-based screening method for agonist selectivity for insulin signaling pathways based on the binding of phosphotyrosine to its specific binding protein, (1-iii) a fluorescent indicator for tyrosine phosphorylation-based insulin signaling pathways, and (1-iv) split luciferase as an optical probe for detecting protein-protein interactions in mammalian cells based on protein splicing; (2) a screening method for antigen-specific IgE using mast cells based on intracellular calcium signaling; (3) a screening method for substrates of multidrug resistance-associated protein (MRP); and (4) fluorescent indicators for cyclic GMP based on cyclic GMP-dependent protein kinase Ialpha and green fluorescent proteins.
- AU Umezawa Yoshio; Ozawa Takeaki; Sato Moritoshi
- SO Analytical sciences: international journal of the Japan Society for Analytical Chemistry, (2002 May) 18 (5) 503-16. Ref: 49 Journal code: 8511078. ISSN: 0910-6340.
- TI Methods of analysis for chemicals that promote/disrupt cellular signaling.
- AB Methods of analysis were presented for chemicals that promote or

disrupt cellular signaling pathways. The developed analytical methods are based not only on receptor binding, but also on the following known molecular-level processes involved in signal transduction along signaling pathways, reconstituted in vitro or taken in part in living cells. The methods were discussed in relation to receptor binding assay and/or bioassay. include: (1) Insulin signaling pathways; (1-i) Chemical selectivity of agonists for insulin signaling pathways based on agonist-induced phosphorylation of a target peptide; (1-ii) An SPR-based screening method for agonist selectivity for insulin signaling pathways based on the binding of phosphotyrosine to its specific binding protein; (1-iii) A fluorescent indicator for tyrosine phosphorylation-based insulin signaling pathways; (2) An optical method for evaluating ion selectivity for calcium signaling pathways in the cell; (3) Assay and screening of chemicals that disrupt cellular signaling pathways, potential endocrine disruptors in particular; (4) Protein conformational changes, and (5) A screening method for antigen-specific IgE using mast cells, based on intracellular calcium signaling.

- AU Umezawa Y.; Ozawa T.; Sato M.
- SO Bulletin of the Chemical Society of Japan, (2002) 75/7 (1423-1433). Refs: 46
  ISSN: 0009-2673 CODEN: BCSJA8
- TI Probing chemical processes in living cells: Applications for assay and screening of chemicals that disrupt cellular signaling pathways.
- Methods of analysis for bioactive substances that are based on cellular signaling pathways are discussed and compared with the binding assay and bioassay. For nondestructive analysis of chemical processes in living cells, we have developed some general methods and new intracellular fluorescent probes for detecting 1) second messengers, cGMP, diacylglycerol and phosphatidylinositol-3,4,5-triphosphate, 2) protein phosphorylation, 3) protein conformational changes, and 4) protein-protein interactions in live cells under a confocal laser microscope. The approaches for the present probe developments are use of fluorescence resonance energy transfer for reporting binding of substrates (analytes) to molecular recognition domains in dual-fluorophore conjugated probe molecules, and use of protein splicing chemistry for detecting protein-protein interactions. Key molecules and steps of cellular signaling pathways were visualized in relevant live cells using developed fluorescent probe molecules. These probes were found of general importance not only for fundamental biology studies, but also for assay and screening methods for chemicals that inhibit or facilitate cellular signaling pathways. Changes in cellular signals were thereby observed in nongenomic pathways of steroid hormones upon treatment of the target cells with steroid hormones and xenoestrogens. This method of analysis appears to be a rational approach to high-throughput prescreening of biohazardous chemicals such as endocrine disrupting chemicals that disrupt these cellular signaling pathways.
- AU Fujita, Hidetomo; Takemura, Miho; Tani, Emi; Nemoto, Kyoko; Yokota, Akiho; Kohchi, Takayuki [Reprint Author]
- SO Plant and Cell Physiology, (July 2003) Vol. 44, No. 7, pp. 735-742. print. ISSN: 0032-0781 (ISSN print).
- TI An Arabidopsis MADS-box protein, AGL24, is specifically bound to and phosphorylated by Meristematic Receptor-Like Kinase (MRLK).
- AB Intercellular signaling mediated by receptor-like kinases (RLKs) is important for diverse processes in plant development, although downstream intracellular signaling pathways remain poorly understood. Proteins interacting directly with RLK were screened for by yeast two-hybrid assay with the kinase domain as bait. A MADS-box protein, AGL24 was identified as a candidate substrate of MRLK (Meristematic Receptor-Like Kinase), which was named for its spatial expression in shoot and root apical meristems in Arabidopsis. The AGL24 protein specifically interacted with, and was phosphorylated by, the MRLK kinase domain in in vitro assays. The simultaneous expression of AGL24 and MRLK in shoot apices during floral transition suggested that the interaction occurs in plants. Using plants constitutively expressing a fusion protein of AGL24 and green fluorescent protein, the

subcellular localization of AGL24 protein was observed exclusively in the nucleus in apical tissues where MRLK was expressed, while AGL24 was localized in both the cytoplasm and the nucleus in tissues where no MRLK expression was detectable. These results suggest that MRLK signaling promotes translocation of AGL24 from the cytoplasm to the nucleus. We propose that the RLK signaling pathway involves phosphorylation of a MADS-box transcription factor.

- AU Witting S.R.; Maiorano J.N.; Davidson W.S.
- SO Journal of Biological Chemistry, (10 Oct 2003) 278/41 (40121-40127).
  Refs: 70
  ISSN: 0021-9258 CODEN: JBCHA3
- TI Ceramide enhances cholesterol efflux to apolipoprotein A-I by increasing the cell surface presence of ATP-binding cassette transporter A1.
- It is widely accepted that functional ATP-binding cassette transporter A1 AB (ABCA1) is critical for the formation of nascent high density lipoprotein particles. However, the cholesterol pool(s) and the cellular signaling processes utilized by the ABCA1-mediated pathway remain unclear. Sphingomyelin maintains a preferential interaction with cholesterol in membranes, and its catabolites, especially ceramide, are potent signaling molecules that could play a role in ABCA1 regulation or function. To study the potential role of ceramide in this process, we treated a variety of cell lines with 20 <SYM109>M C(2)-ceramide and examined apolipoprotein-mediated cholesterol efflux to lipid-free apoA-I. We found that cell lines expressing ABCA1 displayed 2-3-fold increases in cholesterol efflux to apoA-I. Cell lines not expressing ABCA1 were unaffected by ceramide. We further characterized the cholesterol efflux effect in Chinese hamster ovary cells. Ceramide treatment did not cause significant cytotoxicity or apoptosis and did not affect cholesterol efflux to non-apolipoprotein acceptors. Raising endogenous ceramide levels increased cholesterol efflux to apoA-I. Using a cell surface biotinylation method, we found that the total cellular ABCA1 and that at the plasma membrane were increased with ceramide treatment. Also ceramide enhanced the binding of fluorescently labeled apoA-I to Chinese hamster ovary cells. These data suggest that ceramide may increase the plasma membrane content of ABCA1, leading to increased apoA-I binding and cholesterol efflux.
- AU Almholt D.L.C.; Loechel F.; Nielsen S.J.; Krog-Jensen C.; Terry R.; Bjorn S.P.; Pedersen H.C.; Praestegaard M.; Moller S.; Heide M.; Pagliaro L.; Mason A.J.; Butcher S.; Dahl S.W.
- SO Assay and Drug Development Technologies, (2004) 2/1 (7-20).
  Refs: 37
  ISSN: 1540-658X CODEN: ADDTAR
- TI Nuclear export inhibitors and kinase inhibitors identified using a MAPK-activated protein kinase 2 redistribution® screen.
- AB Redistribution® (BioImage® A/S, Soborg, Denmark) is a novel high-throughput screening technology that monitors translocation of specific protein components of intracellular signaling pathways within intact mammalian cells, using green fluorescent protein as a tag. A single Redistribution assay can be used to identify multiple classes of compounds that act at, or upstream of, the level of the protein target used in the primary screening assay. Such compounds may include both conventional and allosteric enzyme inhibitors, as well as protein-protein interaction modulators. We have developed a series of Redistribution assays to discover and characterize compounds that inhibit tumor necrosis factor-<SYM97> biosynthesis via modulation of the p38 mitogen-activated protein kinase (MAPK) pathway. A primary assay was designed to identify low-molecular-weight compounds that inhibit the activation-dependent nuclear export of the p38 kinase substrate MAPK-activated protein kinase 2 (MK2). Hits from the primary screen were categorized, using secondary assays, either as direct inhibitors of MK2 nuclear export, or as inhibitors of the upstream p38 MAPK pathway. Activity profiles are presented for a nuclear export inhibitor, and a compound that structurally and functionally resembles a known p38 kinase inhibitor. These results demonstrate the utility of Redistribution technology as a pathway screening method for the identification of diverse and novel compounds that are active within therapeutically important signaling pathways. . COPYRGT. Mary Ann Liebert, Inc.

Assay and Drug Development Technologies (2004), 2(5), 473-481 CODEN: ADDTAR; ISSN: 1540-658X

A Quantitative Cell-Based High-Content Screening Assay for the Epidermal Growth Factor Receptor-Specific Activation of Mitogen-Activated Protein Kinase

AB The complexity of mitogen-activated protein kinase (MAPK) signaling pathways and their activation by different stimuli makes assaying the activation of particular MAPKs by specific receptors a challenging problem. The multiplexing capability of quant. high-content screening (HCS) assays enables the simultaneous monitoring and correlation, in the same cell, of an MAPK's specific activation with a particular receptor's post-signaling behavior, such as its internalization. We demonstrate a cell-based HCS assay to quantify the epidermal growth factor (EGF) receptor-specific activation of the MAPK ERK. Activation was quantified by measuring immunofluorescently labeled phosphorylated extracellular signal-regulated protein kinases (ERK) in the nucleus. Specificity of ERK activation by the EGF receptor was simultaneously confirmed in the same cell by quant. monitoring fluorescent EGF's internalization and subsequent intracellular degradation Quant. anal. of the temporal behavior of these two activities showed that phosphorylated ERK's accumulation in the nucleus peaked at 5 min before falling to basal levels by 30 min. Cellular accumulation of fluorescent EGF was slower, peaking around 30 min, before being degraded. This assay strategy can serve as a paradigm to study other signaling pathways and their activation by specific receptors. The flexibility and multiplexing capability of HCS assays allow the use of addnl. targets to further qualify the specificity of response by including other MAPKs or receptors, to rule out cross-talk from competing signaling pathways, or to simultaneously monitor toxicity effects of compds. automated, non-subjective, easy-to-use assay procedure provides information rich, quant. results, and demonstrates the potential of the HCS assay approach in deconvolving intracellular signaling pathways.

Kawai Y.; Sato M.; Umezawa Y. ΑU

Analytical Chemistry, (15 Oct 2004) 76/20 (6144-6149). Refs: 29

ISSN: 0003-2700 CODEN: ANCHAM

TISingle color fluorescent indicators of protein phosphorylation for multicolor imaging of intracellular signal flow dynamics.

Existing monitoring methods for protein phosphorylation involved AB in intracellular signal transduction in vivo are exclusively based on fluorescence resonance energy transfer, which needs the measurement of the change in fluorescence intensities at two wavelengths. Therefore, it is difficult to monitor protein phosphorylation together with other related signaling processes, such as second messengers and protein translocation. To overcome this problem, we developed novel fluorescent indicators, each containing a differently colored (cyan and green) single fluorophore. The present indicator is a tandem fusion protein containing a kinase substrate domain, a circularly permuted fluorescent protein (cpFP), and a phosphorylation recognition domain. The cpFP is obtained by dividing a green fluorescent protein mutant (GFP) at residue 144-145 and linking the carboxy and amino portions thereof with a peptide linker. The substrate domain used in this study is a peptide sequence that is phosphorylated by insulin receptor. Phosphorylation of the substrate domain induces its interaction with the phosphorylation recognition domain, which causes a conformational change in the cpFP and a change in its fluorescence. The cyan and green indicators exhibited 10% decrease and 15% increase, respectively, in their fluorescence intensities upon phosphorylation. Using this cyan indicator and GFP-tagged mitogen-activated protein kinase (MAPK), we found that insulin-induced protein phosphorylation occurred immediately upon the addition of insulin, whereas nuclear translocation of MAPK occurred 7 min later. By tailoring the substrate domains and the phosphorylation recognition domains in these cyan and green indicators, the present approach should be applicable to the in vivo analysis of a broad range of protein phosphorylation processes, together with other intracellular signaling processes.

Thastrup, Ole; Petersen Bjorn, Sara; Tullin, Soren; Kasper, Almholt; Scudder, Kurt PCT Int. Appl., 327 pp.

SO

CODEN: PIXXD2

TI Characterizing a cellular response to a stimulus using a lumiphore genetically modified with a signal pathway component

AΒ Cells are genetically modified to express a luminophore, e.g., a modified (F64L, S65T, Y66H) Green Fluorescent Protein (GFP, EGFP) coupled to a component of an intracellular signaling pathway such as a transcription factor, a cGMP- or cAMP-dependent protein kinase, a cyclin-, calmodulin- or phospholipid-dependent or mitogen-activated serine/threonine protein kinase, a tyrosine protein kinase, or a protein phosphatase (e.g. PKA, PKC, Erk, Smad, VASP, actin, p38, Jnk1, PKG, IkappaB, CDK2, Grk5, Zap70, p85, protein-tyrosine phosphatase 1C, Stat5, NFAT, NFkappaB, RhoA, PKB). An influence modulates the intracellular signaling pathway in such a way that the luminophore is being redistributed or translocated with the component in living cells in a manner exptl. determined to be correlated to the degree of the influence. Measurement of redistribution is performed by recording of light intensity, fluorescence lifetime, polarization, wavelength shift, resonance energy transfer, or other properties by an apparatus consisting of e.g. a fluorescence microscope and a CCD camera. stored as digital images are processed to nos. representing the degree of redistribution. The method can be used as a screening program for identifying a compound that modulates a component and is capable of treating a disease related to the function of the component.



Day: Thursday Date: 12/9/2004

Time: 15:13:21

#### **Inventor Name Search**

Enter the **first few letters** of the Inventor's Last Name. Additionally, enter the **first few letters** of the Inventor's First name.

Last Name	First Name	
thastrup	ole	Search

To go back use Back button on your browser toolbar.



Day: Thursday Date: 12/9/2004

Time: 15:13:21

#### **Inventor Name Search**

Enter the **first few letters** of the Inventor's Last Name. Additionally, enter the **first few letters** of the Inventor's First name.

Last Name	First Name	
petersen	sara	Search

To go back use Back button on your browser toolbar.



Day: Thursday Date: 12/9/2004

Time: 15:13:21

### **Inventor Name Search**

Enter the **first few letters** of the Inventor's Last Name. Additionally, enter the **first few letters** of the Inventor's First name.

Last Name	First Name	
bjorn	sara	Search
The second section of the second seco	The comment of the co	

To go back use Back button on your browser toolbar.



Day: Thursday Date: 12/9/2004

Time: 15:13:21

### **Inventor Name Search**

Enter the **first few letters** of the Inventor's Last Name. Additionally, enter the **first few letters** of the Inventor's First name.

Last Name	First Name	
tullin	soren	Search

To go back use Back button on your browser toolbar.



Day: Thursday Date: 12/9/2004

Time: 15:13:21

#### **Inventor Name Search**

Enter the **first few letters** of the Inventor's Last Name. Additionally, enter the **first few letters** of the Inventor's First name.

Last Name	First Name	
almholt	kasper	Search

To go back use Back button on your browser toolbar.



Day: Thursday Date: 12/9/2004

Time: 15:13:21

### **Inventor Name Search**

Enter the **first few letters** of the Inventor's Last Name. Additionally, enter the **first few letters** of the Inventor's First name.

Last Name	First Name	
scudder	kurt	Search

To go back use Back button on your browser toolbar.

Back to  $\underline{PALM} \mid \underline{ASSIGNMENT} \mid \underline{OASIS} \mid Home page$